Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI,TDBD; THES=ASSIGNEE; PLUR=YES; OP=AND			
L12	L6 not L8	17	L12
L11	L8 not L9	20	L11
L10	L9 and (C17.2)	2	L10
L9	L6 and (prodrug or (cytosine adj deaminase))	23	L9
L8	L7 and L6	31	L8
L7	(packaging or (producer adj cell))	289373	L7
L6	L5 and (engraftable or implantable)	48	L6
L5	L3 and L2	143	L5
L4	L3 same L2	16	L4
L3	(brain adj (tumor or cancer)) or (glioblastoma or astrocytoma or gliomas)	12579	L3
L2	((neural adj stem) adj cell) or (neural adj progenitor)	849	L2
L1	Snyder-Evan-Y$.in.	6	L1

END OF SEARCH HISTORY

Status: Path 1 of [Dialog Information Services via Modem]

Status: Initializing TCP/IP using (UseTelnetProto 1 ServiceID pto-dialog)
Trying 31060000009999...Open

DIALOG INFORMATION SERVICES
PLEASE LOGON:
 ******** HHHHHHHH SSSSSSSS?
Status: Signing onto Dialog
 ****
ENTER PASSWORD:
 ******** HHHHHHHH SSSSSSSS? ********
Welcome to DIALOG
Status: Connected

Dialog level 03.03.02D

Last logoff: 30sep03 13:19:40
Logon file001 06oct03 15:13:59
           *** ANNOUNCEMENT ***
                    ***
--File 654 - US published applications from March 15, 2001 to the
present are now online. Please see HELP NEWS 654 for details.

                    ***
--File 581 - The 2003 annual reload of Population Demographics is
complete. Please see Help News581 for details.

                    ***
                    ***
--File 990 - NewsRoom now contains February 2003 to current records.
File 992 - NewsRoom 2003 archive has been newly created and contains
records from January 2003. The oldest months's records roll out of
File 990 and into File 992 on the first weekend of each month.
To search all 2003 records BEGIN 990, 992, or B NEWS2003, a new
OneSearch category.
                    ***
--Connect Time joins DialUnits as pricing options on Dialog.
See HELP CONNECT for information.
                    ***
                    ***
--SourceOne patents are now delivered to your email inbox
as PDF replacing TIFF delivery. See HELP SOURCE1 for more
information.
                    ***
--Important news for public and academic
libraries. See HELP LIBRARY for more information.
                    ***
--Important Notice to Freelance Authors--
See HELP FREELANCE for more information
                    ***
NEW FILES RELEASED
***World News Connection (File 985)
***Dialog NewsRoom - 2003 Archive (File 992)
***TRADEMARKSCAN-Czech Republic (File 680)
***TRADEMARKSCAN-Hungary (File 681)
***TRADEMARKSCAN-Poland (File 682)
                    ***
UPDATING RESUMED
                    ***
RELOADED
***Population Demographics -(File 581)
***CLAIMS Citation (Files 220-222)

REMOVED

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                    ***

     >>> Enter BEGIN HOMEBASE for Dialog Announcements <<<
     >>>    of new databases, price changes, etc.      <<<
                    ****
KWIC is set to 50.
HILIGHT set on as '*'

File   1:ERIC  1966-2003/Oct 06
       (c) format only 2003 The Dialog Corporation

      Set  Items  Description
      ---  -----  -----------
Cost is in DialUnits
?b 155, 159, 5, 73
       06oct03 15:14:17 User259876 Session D553.1
            $0.32    0.090 DialUnits File1
     $0.32  Estimated cost File1
     $0.07  TELNET
     $0.39  Estimated cost this search
     $0.39  Estimated total session cost   0.090 DialUnits


SYSTEM:OS  - DIALOG OneSearch
  File 155:MEDLINE(R)  1966-2003/Oct W1
        (c) format only 2003 The Dialog Corp.
*File 155: Medline has been reloaded and accession numbers have
changed.  Please see HELP NEWS 155.
  File 159:Cancerlit  1975-2002/Oct
        (c) format only 2002 Dialog Corporation
*File 159: Cancerlit ceases updating with immediate effect.
Please see HELP NEWS.
  File   5:Biosis Previews(R)  1969-2003/Sep W4
        (c) 2003 BIOSIS
  File  73:EMBASE  1974-2003/Sep W4
        (c) 2003 Elsevier Science B.V.

      Set  Items  Description
      ---  -----  -----------
?s ((neural (w) stem (w) cells) or (neural (w) progenitors))
          931871  NEURAL
          363245  STEM
         5065393  CELLS
            2227  NEURAL(W)STEM(W)CELLS
          931871  NEURAL
           42151  PROGENITORS
             664  NEURAL(W)PROGENITORS
      S1    2818  ((NEURAL (W) STEM (W) CELLS) OR (NEURAL (W) PROGENITORS))
?s (brain (w) (tumor or cancer)) or (glioblastoma or astrocytoma or gliomas)
         1604515  BRAIN
         2171259  TUMOR
         2299044  CANCER
           42556  BRAIN(W)(TUMOR OR CANCER)
           33198  GLIOBLASTOMA
           34942  ASTROCYTOMA
           28803  GLIOMAS
      S2  107952  (BRAIN (W) (TUMOR OR CANCER)) OR (GLIOBLASTOMA OR
                  ASTROCYTOMA OR GLIOMAS)
?s s1 (s) s2
            2818  S1
          107952  S2
      S3      64  S1 (S) S2
?rd
...examined 50 records  (50)
...completed examining records
      S4      26  RD (unique items)
?t s4/3,k/all
```

4/3,K/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15319123 22832824 PMID: 12952304
Neural stem cell biology may be well suited for improving brain tumor therapies.
Yip Stephen; Aboody Karen S; Burns Michael; Imitola Jaime; Boockvar John A; Allport Jennifer; Park Kook In; Teng Yang D; Lachyankar Mahesh; McIntosh Tracy; O'Rourke Donald M; Khoury Samia; Weissleder Ralph; Black Peter M; Weiss William; Snyder Evan Y
The Burnham Institute, Program in Developmental & Regenerative Cell Biology, La Jolla, California 92037, USA.
Cancer journal (Sudbury, Mass.) (United States) May-Jun 2003, 9 (3) p189-204, ISSN 1528-9117 Journal Code: 100931981
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: In Process

Neural *stem* *cells* (NSCs) are capable of tremendous migratory potential to areas of pathology in the central nervous system. When implanted into a diseased or injured nervous system...

... various central nervous system pathologies, especially brain tumors. Brain tumors are generally difficult to treat because of the unique location of the lesions. In primary *gliomas*, the extensive infiltrative nature of the tumor cells presents a challenge for their effective and total eradication, hence the high rate of treatment failure and...

... of NSCs to "home in" on tumor cells followed by the delivery of a desired gene product makes the NSC a very promising agent in *brain* *tumor* therapy. Cytolytic viruses and genes coding for anti-tumor cytokines, pro-drug converting enzymes, and various neurotrophic factors have all been engineered into engraftable NSCs...

4/3,K/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15132347 22586190 PMID: 12700036
Mechanisms of tumor cell invasion and angiogenesis in the central nervous system.
Visted Therese; Enger Per Oyvind; Lund-Johansen Morten; Bjerkvig Rolf
Department of Anatomy and Cell Biology, University of Bergen, Aarstadveien 19, N-5009, Bergen, Norway.
Frontiers in bioscience - a journal and virtual library (United States) May 1 2003, 8 pe289-304, ISSN 1093-4715 Journal Code: 9709506
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: In Process

Despite extensive progress in characterizing the genetic events involved in the development of *gliomas* , the cellular origin and the defined molecular mechanisms that lead to their occurrence are still unclear. It is known that tumours are of monoclonal origin. This is contrasted by the fact that *gliomas* frequently express features of different glial cell lineages. With the identification of pluripotent *neural* *stem* *cells* and the growth factors that control neural cell development, we are now making early inroads towards understanding glial cell migration as well as the neural cell plasticity within the adult central nervous system (CNS). *Gliomas* share several fetal antigens with immature brain cells. It is therefore tempting to speculate that the migration of neural precursor cells actually represents the normal counterpart of glioma cell migration.

The migratory behavior of gliomas* may be due to a predetermined interplay between normal brain tissue and the migrating cells, where the brain represents a permissive tissue for guiding cells with certain phenotypic traits to migrate along specific anatomical structures. Malignant progression is also accompanied by extensive angiogenesis which is especially prominent in *glioblastoma* multiforme (GBM). For cell proliferation to take place, several cell signaling cues mediated by specific growth factors are shared between the glioma cells and the...

4/3,K/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15131666 22344464 PMID: 12456351
Targeted therapy of brain tumors utilizing neural stem and progenitor cells.
Burns Michael J; Weiss William
University of California San Francisco, Department of Neurology, Box 0114, San Francisco, CA 94143, USA. brains@itsa.ucsf.edu
Frontiers in bioscience - a journal and virtual library (United States) Jan 1 2003, 8 pe228-34, ISSN 1093-4715 Journal Code: 9709506
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: In Process

... 3. The pluripotentcy of neural stem and progenitor cells allows them to engraft and replace damaged tissues in the CNS. These properties suggest that transplanted *neural* *stem* *cells* might be used to deliver molecular therapy to diseased regions of the nervous system, and to regenerate lost tissues. One of the greatest challenges and...

... cells to migrate extensively into normal brain parenchyma in part underlies the lethal nature of these tumors. A better understanding of the mechanisms by which *neural* *stem* *cells* migrate to specific sources of injury may allow us to harness these cells as vehicles for delivery of molecular therapies to impact survival in patients with recalcitrant *gliomas*.

4/3,K/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15109425 22649205 PMID: 12762886
The neurosurgeon as local oncologist: cellular and molecular neurosurgery in malignant glioma therapy.
Dunn Ian F; Black Peter McL
Brain Tumor Laboratories and Department of Neurosurgery, Brigham and Women's Hospital, Boston, MA, USA.
Neurosurgery (United States) Jun 2003, 52 (6) p1411-22; discussion 1422-4, ISSN 0148-396X Journal Code: 7802914
Document type: Journal Article; Review; Review, Academic
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Malignant *gliomas* are among the most challenging of all cancers to treat successfully, being characterized not only by aggressive proliferation and expansion but also by inexorable tumor...

... and minimize systemic toxicity. Exciting local-therapy approaches have been proposed for these devastating tumors. In this review, we discuss the potential applications of bioreactors, *neural* *stem* *cells* , immunotherapies, biodegradable polymers, and convection-enhanced drug delivery in the treatment of malignant *gliomas*. These approaches are at

different stages of readiness for application in clinical neurosurgery, and their eventual effects on the morbidity and mortality rates of *gliomas* among human patients are difficult to ascertain from successes in animal models. Nevertheless, we are entering an exciting era of "nanoneurosurgery," in which molecular therapies...

...of neuro-oncological disease. The potential to deploy any of a number of eloquently devised molecular therapies may provide renewed hope for neurosurgeons treating malignant *gliomas*.

4/3,K/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15095918 22445254 PMID: 12558076
Signaling pathways regulating gliomagenesis.
Konopka G; Bonni A
Department of Pathology and Program in Neuroscience, Harvard Medical School, Boston, MA 02115, USA.
Current molecular medicine (Netherlands) Feb 2003, 3 (1) p73-84, ISSN 1566-5240 Journal Code: 101093076
Contract/Grant No.: R01-NS41021-01; NS; NINDS
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

...represent the most common primary tumors of the brain. Despite efforts to improve the treatment of astrocytomas, these tumors and in particular the high-grade *astrocytoma* termed *glioblastoma* multiforme still carry a poor prognosis. In recent years, there has been an intensive effort to gain an understanding of the cellular and molecular mechanisms...

... the cell-of-origin of astrocytomas has been investigated. Genetic modeling of astrocytomas in mice suggests that neuroepithelial precursor cells represent preferred cellular substrates of *gliomas* or that either astrocytes or precursor cells constitute potential cells-of-origin of astrocytomas. During normal brain development, neuroepithelial precursor cells, including *neural* *stem* *cells*, differentiate into astrocytes. As the mechanisms that control gliogenesis during normal brain development become better understood, it will be important to determine if deregulation of...

4/3,K/6 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14905499 22620256 PMID: 12735302
The neural progenitor-restricted isoform of the MARK4 gene in 19q13.2 is upregulated in human gliomas and overexpressed in a subset of glioblastoma cell lines.
Beghini Alessandro; Magnani Ivana; Roversi Gaia; Piepoli Tiziana; Di Terlizzi Simona; Moroni Ramona F; Pollo Bianca; Fuhrman Conti Anna M; Cowell John K; Finocchiaro Gaetano; Larizza Lidia
Department of Biology and Genetics, University of Milan, via Viotti 3/5, 20133 Milan, Italy. alessandro.beghini@unimi.it
Oncogene (England) May 1 2003, 22 (17) p2581-91, ISSN 0950-9232 Journal Code: 8711562
Contract/Grant No.: CA76457; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... in glial neoplasms, suggesting that this region harbors at least one

gene involved in gl[redacted]agenesis. Following our pre[redacted]us studies on structural 19q chromosome rearrangements in *gliomas*, we have undertaken a detailed FISH analysis of the breakpoints and identified a 19q13.2 intrachromosomal amplification of the MAP/microtubule affinity-regulating kinase 4 (MARK4) gene in three primary *glioblastoma* cell lines. Recent data suggest that this gene is involved in the Wnt-signaling pathway. We observed that the expression of the alternatively spliced MARK4L isoform is upregulated in both fresh and cultured *gliomas* and overexpressed in all of the above three *glioblastoma* cell lines. Interestingly, we also found that MARK4L expression is restricted to undifferentiated neural progenitor cells or proliferating glial precursor cells, whereas its expression is downregulated during glial differentiation. Perturbation of expression using antisense oligonucleotides against MARK4 in *glioblastoma* cell lines, consistently induced a decreased proliferation of tumor cells. Taken together, these data show that MARK4, which is normally expressed in *neural* *progenitors*, is re-expressed in *gliomas* and may become a key target of intrachromosomal amplification upon 19q rearrangements.

4/3,K/7 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14379125 22387061 PMID: 12499252
Induction of glioblastoma apoptosis using neural stem cell-mediated delivery of tumor necrosis factor-related apoptosis-inducing ligand.
Ehtesham Moneeb; Kabos Peter; Gutierrez Mervin A R; Chung Nancy H C; Griffith Thomas S; Black Keith L; Yu John S; et al
Maxine Dunitz Neurosurgical Institute, Cedars-Sinai Medical Center, Los Angeles, California 90048, USA.
Cancer research (United States) Dec 15 2002, 62 (24) p7170-4, ISSN 0008-5472 Journal Code: 2984705R
Contract/Grant No.: NS02232; NS; NINDS; +
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Current therapies for *gliomas* fail to address their highly infiltrative nature. Standard treatments often leave behind microscopic neoplastic reservoirs, resulting in eventual tumor recurrence. *Neural* *stem* *cells* (NSCs) are capable of tracking disseminating glioma cells. To exploit this tropism to develop a therapeutic strategy that targeted tumor satellites, we inoculated human *glioblastoma* xenografts with tumor necrosis factor-related apoptosis-inducing ligand-secreting NSCs. This resulted in the dramatic induction of apoptosis in treated tumors and tumor satellites ...

4/3,K/8 (Item 8 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10114702 22081554 PMID: 12086863
Epidermal growth factor receptor and Ink4a/Arf: convergent mechanisms governing terminal differentiation and transformation along the neural stem cell to astrocyte axis.
Bachoo Robert M; Maher Elizabeth A; Ligon Keith L; Sharpless Norman E; Chan Suzanne S; You Mingjian James; Tang Yi; DeFrances Jessica; Stover Elizabeth; Weissleder Ralph; Rowitch David H; Louis David N; DePinho Ronald A
Center for Neuro-Oncology, Boston, Massachusetts 02115, USA.
Cancer cell (United States) Apr 2002, 1 (3) p269-77, ISSN 1535-6108 Journal Code: 101130617
Contract/Grant No.: P30; PHS
Document type: Journal Article
Languages: ENGLISH

Main Citation Owner: N
Record type: Completed

Ink4a/Arf inactivation and epidermal growth factor receptor (EGFR) activation are signature lesions in high-grade *gliomas* . How these mutations mediate the biological features of these tumors is poorly understood. Here, we demonstrate that combined loss of p16(INK4a) and p19(ARF), but not of p53, p16(INK4a), or p19(ARF), enables astrocyte dedifferentiation in response to EGFR activation. Moreover, transduction of Ink4a/Arf(-/-) *neural* *stem* *cells* (NSCs) or astrocytes with constitutively active EGFR induces a common high-grade glioma phenotype. These findings identify NSCs and astrocytes as equally permissive compartments for...

... differentiation. These data support the view that dysregulation of specific genetic pathways, rather than cell-of-origin, dictates the emergence and phenotype of high-grade *gliomas*.

4/3,K/9 (Item 9 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09767288 21572158 PMID: 11716071
Glioma migration: clues from the biology of neural progenitor cells and embryonic CNS cell migration.
Dirks P B
Division of Neurosurgery, Hospital for Sick Children, University of Toronto, Ontario, Canada. peter.dirks@sickkids.on.ca
Journal of neuro-oncology (Netherlands) Jun 2001, 53 (2) p203-12, ISSN 0167-594X Journal Code: 8309335
Document type: Journal Article; Review; Review, Academic
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Neural *stem* *cells* have recently come to the forefront in neurobiology because of the possibilities for CNS repair by transplantation. Further understanding of the biology of these cells...

... progenitor cell. An understanding of the normal migratory ability of these cells is also likely to have a very important impact on the knowledge of *brain* *tumor* invasion.

4/3,K/10 (Item 10 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09740617 21542430 PMID: 11686236
Musashi1, an evolutionarily conserved neural RNA-binding protein, is a versatile marker of human glioma cells in determining their cellular origin, malignancy, and proliferative activity.
Kanemura Y; Mori K; Sakakibara S; Fujikawa H; Hayashi H; Nakano A; Matsumoto T; Tamura K; Imai T; Ohnishi T; Fushiki S; Nakamura Y; Yamasaki M ; Okano H; Arita N
Institute for Clinical Research, Osaka National Hospital, Japan.
Differentiation; research in biological diversity (Germany) Sep 2001, 68 (2-3) p141-52, ISSN 0301-4681 Journal Code: 0401650
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... phenotypic markers that are specific to the cells from which they originated. A neural RNA-binding protein, Musashi1, is an evolutionarily well-conserved marker for *neural* *stem* *cells*/ progenitor cells. To

examine the origin of [redacted]iomas*, we examined the expres[redacted]n of the human Musashil homolog, MSI1, in human glioma tissues and in n[redacted]mal human adult and fetal brains. As we had...
... with proliferative activity, as estimated by an MIB-1 staining index. Our results indicate that MSI1 is an excellent marker for neural progenitor cells including *neural* *stem* *cells* in normal human brains. Furthermore, the expression of MSI1 correlates well with the immature nature as well as the malignancy of tumor cells in human *gliomas*. Thus, we expect the analysis of MSI1 expression to contribute to the understanding of the cellular origin and biology of human *gliomas*.

4/3,K/11 (Item 11 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09594374 21378377 PMID: 11485986
PDGF autocrine stimulation dedifferentiates cultured astrocytes and induces oligodendrogliomas and oligoastrocytomas from neural progenitors and astrocytes in vivo.
Dai C; Celestino J C; Okada Y; Louis D N; Fuller G N; Holland E C
Department of Neurosurgery, Memorial Sloan-Kettering Cancer Center, New York, New York 10021, USA.
Genes & development (United States) Aug 1 2001, 15 (15) p1913-25, ISSN 0890-9369 Journal Code: 8711660
Contract/Grant No.: UO1CA894314-1; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... type-specific gene transfer we investigated the functions of PDGF autocrine signaling in gliomagenesis by transferring the overexpression of PDGF-B into either nestin-expressing *neural* *progenitors* or glial fibrillary acidic protein (GFAP)-expressing astrocytes both in cell culture and in vivo. In cultured astrocytes, overexpression of PDGF-B caused significant increase in proliferation rate of both astrocytes and *neural* *progenitors* . Furthermore, PDGF gene transfer converted cultured astrocytes into cells with morphologic and gene expression characteristics of glial precursors. In vivo, gene transfer of PDGF to *neural* *progenitors* induced the formation of oligodendrogliomas in about 60% of mice by 12 wk of age; PDGF transfer to astrocytes induced the formation of either oligodendrogliomas or mixed oligoastrocytomas in about 40% of mice in the same time period. Loss of Ink4a-Arf, a mutation frequently found in high-grade human *gliomas* , resulted in shortened latency and enhanced malignancy of *gliomas*. The highest percentage of PDGF-induced malignant *gliomas* arose from of Ink4a-Arf null progenitor cells. These data suggest that chronic autocrine PDGF signaling can promote a proliferating population of glial precursors and...

4/3,K/12 (Item 12 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09492164 21268762 PMID: 11375055
HIV type 1 Nef promotes neoplastic transformation of immortalized neural cells.
Kramer-Hammerle S; Kohleisen B; Hohenadl C; Shumay E; Becker I; Erfle V; Schmidt J
Institute of Molecular Virology, GSF-National Research Center for Environment and Health, D-85764 Neuherberg, Germany.
AIDS research and human retroviruses (United States) May 1 2001, 17 (7) p597-602, ISSN 0889-2229 Journal Code: 8709376
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM

Record type: Completed

...1(Bru)-nef in these cells induced a transformed phenotype and enhanced cell growth in soft agar. Further experiments using previously established nef-expressing human *astrocytoma* cell lines as well as nef-expressing murine fibroblasts suggested a brain cell-specific transforming activity of Nef. After implantation into syngeneic or nude mice both murine and human nef-expressing CNS-derived cells induced tumor development. Interestingly, human *astrocytoma* cells expressing a Nef mutant carrying a disrupted SH3-binding motif involved in protein-protein interactions failed to induce tumor formation. These in vivo data suggest that Nef promotes neoplastic transformation of immortalized murine *neural* *stem* *cells* and enhances malignancy of low-tumorigenic human *astrocytoma* cells. Nef may therefore be involved in the development of AIDS-associated brain tumors.

4/3,K/13 (Item 13 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09487019 21263394 PMID: 11370160
Perspectives on *brain* *tumor* formation involving macrophages, glia, and *neural* *stem* *cells*.
Seyfried T N
Department of Biology, Boston College, Chestnut Hill, MA 02167-3811, USA. thomas.seyfried@bc.edu
Perspectives in biology and medicine (United States) Spring 2001, 44 (2) p263-82, ISSN 0031-5982 Journal Code: 0401132
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Perspectives on *brain* *tumor* formation involving macrophages, glia, and *neural* *stem* *cells*.
The incidence of brain tumors is rising in children and the elderly, but little is known about the mechanisms underlying *brain* *tumor* initiation and progression. In the 1940s, Zimmerman and coworkers exploited the tumor-promoting potential of polycyclic hydrocarbons to produce *brain* *tumor* models in adult mice that simulated the neuropathology of human brain tumors. Based on these early findings and on recent neurobiological studies of stem cells, I propose that crystalline carcinogenic pellets surgically implanted in the central nervous system establish over time a microenvironment that fosters proliferation and genetic damage in *neural* *stem* *cells* and their progenitors. Moreover, activated glia (microglia and astrocytes) and recruited macrophages mediate these processes. Gradually local tissue fields, which normally restrict stem cell proliferation...

... disorganized, leading to further stem cell proliferation, genetic damage, and eventual neoplasia. Depending on age, location, and the state of glial/macrophage activation, the resulting *brain* *tumor* may resemble transformed *neural* *progenitors* aborted in more or less differentiated states. This hypothesis integrates the general mechanisms by which *neural* *stem* *cells* , glia, and macrophages orchestrate the initiation and progression of *brain* *cancer*. Also discussed are implications of these concepts for the diagnosis and therapy of human brain tumors.

4/3,K/14 (Item 14 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09394584 21159785 PMID: 11259117
Selective cell-cycle arrest and induction of apoptosis in proliferating neural cells by ganglioside GM3.
Nakatsuji Y; Miller R H

Department of Neurosciences, Case Western Reserve University School of Medicine, Cleveland, Ohio 44106, USA.
Experimental neurology (United States) Apr 2001, 168 (2) p290-9, ISSN 0014-4886 Journal Code: 0370712
Contract/Grant No.: NS 30800; NS; NINDS; NS 36674; NS; NINDS
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... and differentiation. Here we show that the simple ganglioside GM3 selectively inhibits the proliferation of and induces apoptosis of actively dividing astrocyte precursors and other *neural* *progenitors* . The inhibition of astrocyte precursor proliferation by GM3 appears to be mediated in part by the cyclin-dependent kinase (Cdk) inhibitor p27(Kip1). During neonatal...
... GM3. These data suggest that GM3, a simple ganglioside, may regulate cell proliferation and death in the CNS and as such may have potential for *brain* *tumor* therapy.

4/3,K/15 (Item 15 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09214291 20524089 PMID: 11070094
From the cover: *neural* *stem* *cells* display extensive tropism for pathology in adult brain: evidence from intracranial *gliomas*.
Aboody K S; Brown A; Rainov N G; Bower K A; Liu S; Yang W; Small J E; Herrlinger U; Ourednik V; Black P M; Breakefield X O; Snyder E Y
Departments of Neurology, Pediatrics, and Neurosurgery, Children's Hospital, Boston, MA, USA.
Proceedings of the National Academy of Sciences of the United States of America (UNITED STATES) Nov 7 2000, 97 (23) p12846-51, ISSN 0027-8424 Journal Code: 7505876
Contract/Grant No.: CA69246; CA; NCI; CA86768; CA; NCI; HD07466; HD; NICHD; +
Comment in Proc Natl Acad Sci U S A. 2000 Nov 7;97(23) 12391-2; Comment in PMID 11070072; Comment in Proc Natl Acad Sci U S A. 2000 Nov 7;97(23):12393-5; Comment in PMID 11070073; Erratum in Proc Natl Acad Sci U S A 2001 Jan 16;98(2):777
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
From the cover: *neural* *stem* *cells* display extensive tropism for pathology in adult brain: evidence from intracranial *gliomas*.
... expand, infiltrate surrounding tissue, and migrate widely into normal brain, usually rendering them "elusive" to effective resection, irradiation, chemotherapy, or gene therapy. We demonstrate that *neural* *stem* *cells* (NSCs), when implanted into experimental intracranial *gliomas* in vivo in adult rodents, distribute themselves quickly and extensively throughout the tumor bed and migrate uniquely in juxtaposition to widely expanding and aggressively advancing...

4/3,K/16 (Item 16 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09214206 20524003 PMID: 11070073
Can *neural* *stem* *cells* be used to track down and destroy migratory *brain* *tumor* cells while also providing a means of repairing tumor-associated damage?
Noble M
Center for Cancer Biology, University of Rochester Medical Center, 601 Elmwood Avenue, Box 633, Rochester, NY 14642, USA.

Proceedings of the National Academy of Sciences of the United States of America (UNITED STATES) Nov 7 2000, 97 (23) p12393-5, ISSN 0027-8424 Journal Code: 7505876
Comment on Proc Natl Acad Sci U S A. 2000 Nov 7;97(23) 12846-51; Comment on PMID 11070094
Document type: Comment; Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Can *neural* *stem* *cells* be used to track down and destroy migratory *brain* *tumor* cells while also providing a means of repairing tumor-associated damage?

4/3,K/17 (Item 17 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08973794 20264368 PMID: 10802656
Combined activation of Ras and Akt in *neural* *progenitors* induces *glioblastoma* formation in mice.
Holland E C; Celestino J; Dai C; Schaefer L; Sawaya R E; Fuller G N
Department of Neurosurgery, MD Anderson Cancer Center, Houston, Texas, USA. eholland@notes.mdacc.tmc.edu
Nature genetics (UNITED STATES) May 2000, 25 (1) p55-7, ISSN 1061-4036 Journal Code: 9216904
Contract/Grant No.: CA16672; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Combined activation of Ras and Akt in *neural* *progenitors* induces *glioblastoma* formation in mice.
... are sufficient to induce GBM formation. Here we transfer, in a tissue-specific manner, genes encoding activated forms of Ras and Akt to astrocytes and *neural* *progenitors* in mice. We found that although neither activated Ras nor Akt alone is sufficient to induce GBM formation, the combination of activated Ras and Akt induces high-grade *gliomas* with the histological features of human GBMs. These tumours appear to arise after gene transfer to *neural* *progenitors*, but not after transfer to differentiated astrocytes. Increased activity of RAS is found in many human GBMs (ref. 11), and we show here that Akt...

4/3,K/18 (Item 18 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08918912 20207091 PMID: 10742153
Gene therapy of experimental brain tumors using neural progenitor cells.
Benedetti S; Pirola B; Pollo B; Magrassi L; Bruzzone M G; Rigamonti D; Galli R; Selleri S; Di Meco F; De Fraja C; Vescovi A; Cattaneo E; Finocchiaro G
Istituto Nazionale Neurologico Besta, via Celoria 11, 20133 Milano, Italy.
Nature medicine (UNITED STATES) Apr 2000, 6 (4) p447-50, ISSN 1078-8956 Journal Code: 9502015
Comment in Nat Med. 2000 Apr;6(4) 369-70; Comment in PMID 10742131
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... tumors, have a very poor prognosis. Gene therapy of glioblastomas is limited by the short survival of viral vectors and by their difficulty in

reaching *glioblastoma* cells infiltrating the brain parenchyma. Neural stem/progenitor cells can be engineered to produce therapeutic molecules and have the potential to overcome these limitations because...
... labeled progenitor cells several weeks after the injection. These findings support a new approach for gene therapy of brain tumors, based on the grafting of *neural* *stem* *cells* producing therapeutic molecules.

4/3,K/19 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14428391 BIOSIS NO.: 200300422420
Characterization of neural stem cells derived from human pediatric brain tumors.
AUTHOR: Hemmati Houman D(a); Nakano Ichiro; Masterman-Smith Michael; Lowry Alexandra; Bababeygy Simon; Rafii Benjamin; Minera Miguel; Lazareff Jorge ; Kornblum Harley I; Bronner-Fraser Marianne
AUTHOR ADDRESS: (a)Division of Biology, California Institute of Technology, UCLA School of Medicine, Pasadena, CA, USA**USA
JOURNAL: Developmental Biology 259 (2):p533 July 15 2003 2003
MEDIUM: print
CONFERENCE/MEETING: 62nd Annual Meeting of the Society for Developmental Biology held Jointly with the International Society of Developmental Biologists Boston, MA, USA July 30-August 03, 2003
SPONSOR: International Society of Developmental Biologists
ISSN: 0012-1606
RECORD TYPE: Citation
LANGUAGE: English

DESCRIPTORS:
ORGANISMS: PARTS ETC: *neural* *stem* *cells*--...
...characterization, in-vitro culture, nervous system, pediatric *brain* *tumor* source, tumor development role

4/3,K/20 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14289221 BIOSIS NO.: 200300283250
BRAIN IRRADIATION ENHANCES SURVIVAL OF NEURAL PROGENITOR ALLOGRAFTS TRANSPLANTED INTO NORMAL AND TUMOR-BEARING BRAINS.
AUTHOR: Stauffer W R(a); Fellows-Mayle W(a); Niranjan A(a); Tyler-Kabara E (a); Hong C S; Glorioso J; Gobbel G T(a)
AUTHOR ADDRESS: (a)Dept. Neurological Surgery, University of Pittsburgh, Pittsburgh, PA, USA**USA
JOURNAL: Society for Neuroscience Abstract Viewer and Itinerary Planner 2002pAbstract No 23714 2002
MEDIUM: cd-rom
CONFERENCE/MEETING: 32nd Annual Meeting of the Society for Neuroscience Orlando, Florida, USA November 02-07, 2002
SPONSOR: Society for Neuroscience
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The ability to isolate and cultivate *neural* *progenitors* has engendered novel approaches to replace cells lost due to injury and to deliver gene products to enhance *brain* *tumor* treatment. The goal of our study was to identify the factors that enhance survival of *neural* *progenitors* following transplantation. *Neural* *progenitors* isolated from adult rat (Fisher 344) or mouse (C57BL/6) brain were transplanted into normal or tumor-bearing (RG2 glioma tumor) rat (Fisher 344) brain...

4/3,K/21 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14273787 BIOSIS NO.: 200300267816
A NOVEL ASSAY OF STEM CELLS IN PEDIATRIC BRAIN TUMOURS.
AUTHOR: Singh S K(a); Clarke I D(a); Bonn V E(a); Becker L E(a); Dirks P B (a)
AUTHOR ADDRESS: (a)Labatt Brain Tumor Research Centre, The Hospital for Sick Children, Toronto, ON, Canada**Canada
JOURNAL: Society for Neuroscience Abstract Viewer and Itinerary Planner 2002pAbstract No 712 2002
MEDIUM: cd-rom
CONFERENCE/MEETING: 32nd Annual Meeting of the Society for Neuroscience Orlando, Florida, USA November 02-07, 2002
SPONSOR: Society for Neuroscience
RECORD TYPE: Abstract
LANGUAGE: English

...ABSTRACT: in the brain may be the cause of childhood brain tumors. The purpose of this study is to use techniques developed for the isolation of *neural* *stem* *cells*, and apply them to the characterization of stem cell populations in brain tumors. We apply a panel of neural stem cell markers (including CD-133...
...Based on our data, we show that brain tumors contain cells with stem cell properties, and that CD-133 may serve as a marker of *brain* *tumor* stem cells.

4/3,K/22 (Item 4 from file: 5)

12800498 BIOSIS NO.: 200100007647
***Neural* *stem* *cells* display extensive tropism for pathology in adult brain: Evidence from intracranial *gliomas*.**
AUTHOR: Aboody Karen S; Brown Alice; Rainov Nikolai G; Bower Kate A; Liu Shaoxiong; Yang Wendy; Small Juan E; Herrlinger Ulrich; Ourednik Vaclav; Black Peter McL; Breakefield Xandra O; Snyder Evan Y(a)
AUTHOR ADDRESS: (a)Departments of Neurology, Pediatrics, and Neurosurgery, Children's Hospital, Harvard Medical School, Boston, MA, 02115: Snyder@A1.TCH.Harvard.edu**USA
JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 97 (23):p12846-12851 November 7, 2000
MEDIUM: print
ISSN: 0027-8424
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

***Neural* *stem* *cells* display extensive tropism for pathology in adult brain: Evidence from intracranial *gliomas*.**
...ABSTRACT: expand, infiltrate surrounding tissue, and migrate widely into normal brain, usually rendering them "elusive" to effective resection, irradiation, chemotherapy, or gene therapy. We demonstrate that *neural* *stem* *cells* (NSCs), when implanted into experimental intracranial *gliomas* in vivo in adult rodents, distribute themselves quickly and extensively throughout the tumor bed and migrate uniquely in juxtaposition to widely expanding and aggressively advancing...

4/3,K/23 (Item 5 from file: 5)

12307437 BIOSIS NO.: 200000065304
A novel platform for gene therapy against brain tumors: Foreign gene expressing *neural* *stem* *cells* (NSCs) display tropism for

intracranial *gliomas*
AUTHOR: Aboody Karen S(a); Rainov Nikolai G; Bower Kate A(a); Small Juan E (a); Liu Shaoxiong(a); Brown Alice; McL Black Peter; Breakefield Xandra O ; Snyder Evan Y(a)
AUTHOR ADDRESS: (a)Depts of Neurology, Pediatrics, and Neurosurgery, Children's Hospital, Harvard Med. School, Boston, MA**USA
JOURNAL: Society for Neuroscience Abstracts 25 (1-2):p246 1999
CONFERENCE/MEETING: 29th Annual Meeting of the Society for Neuroscience, Part 1 Miami Beach, Florida, USA October 23-28, 1999
SPONSOR: The Society for Neuroscience
ISSN: 0190-5295
RECORD TYPE: Citation
LANGUAGE: English

A novel platform for gene therapy against brain tumors: Foreign gene expressing *neural* *stem* *cells* (NSCs) display tropism for intracranial *gliomas*.

4/3,K/24 (Item 1 from file: 73)

12236428 EMBASE No: 2003349304
The potential of stem cells for the treatment of brain tumors and globoid cell leukodystrophy
Tunici P.; Pellegatta S.; Finocchiaro G.
P. Tunici, Istituto Nazionale Neurologico Besta, U. of Neuro-Oncology/Gene Therapy, Milan Italy
Cytotechnology (CYTOTECHNOLOGY) (Netherlands) 2003, 41/2-3 (93-101)
CODEN: CYTOE ISSN: 0920-9069
DOCUMENT TYPE: Journal ; Conference Paper
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 60

...their use for brain repair after stroke (Savitz et al., 2002). In this review we will focus on the potential of stem cell treatments for *glioblastoma* multiforme (Holland, 2000), the most aggressive primary *brain* *tumor*, and globoid cell leukodystrophy (Krabbe disease), a metabolic disorder of the white matter (Berger et al., 2001). These two diseases may offer a paradigm of...

...cell approach may offer in term of treatment, alone or in combination with other therapeutic approaches. Two kinds of stem cells will be considered here: *neural* *stem* *cells* and hematopoietic stem cells, both obtained after birth. The review will focus on experimental models, with an eye on clinical perspectives.

4/3,K/25 (Item 2 from file: 73)

11056792 EMBASE No: 2000407577
Can *neural* *stem* *cells* be used to track down and destroy migratory *brain* *tumor* cells while also providing a means of tumor-associated damage?
Noble M.
M. Noble, Center for Cancer Biology, Univ. of Rochester Medical Center, Box 633, 601 Elmwood Avenue, Rochester, NY 14642 United States
Proceedings of the National Academy of Sciences of the United States of America (PROC. NATL. ACAD. SCI. U. S. A.) (United States) 2000, 97/23 (12393-12395)
CODEN: PNASA ISSN: 0027-8424
DOCUMENT TYPE: Journal ; Conference Paper
LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 15

Can *neural* *stem* *cells* be used to track down and destroy migratory *brain* *tumor* cells while also providing a means of tumor-associated damage?

4/3,K/26 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE
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11001397 EMBASE No: 2001042129
Erratum: *Neural* *stem* *cells* display extensive tropism for pathology in adult brain: Evidence from intracranial *gliomas* (Proceedings of the National Academy of Sciences of USA (November 7, 2000) 97 (12846-12851))
Aboody K.S.; Brown A.; Rainov N.G.; Bower K.A.; Liu S.; Yang W.; Small J.E.; Herrlinger U.; Ourednik V.; Black P. McL.; Breakefield X.O.; Snyder E.Y.
Proceedings of the National Academy of Sciences of the United States of America (PROC. NATL. ACAD. SCI. U. S. A.) (United States) 16 JAN 2001, 98/2 (777)
CODEN: PNASA ISSN: 0027-8424
DOCUMENT TYPE: Journal ; Erratum
LANGUAGE: ENGLISH

Erratum: *Neural* *stem* *cells* display extensive tropism for pathology in adult brain: Evidence from intracranial *gliomas* (Proceedings of the National Academy of Sciences of USA (November 7, 2000) 97 (12846-12851))
?ds

Set	Items	Description
S1	2818	((NEURAL (W) STEM (W) CELLS) OR (NEURAL (W) PROGENITORS))
S2	107952	(BRAIN (W) (TUMOR OR CANCER)) OR (GLIOBLASTOMA OR ASTROCYT-OMA OR GLIOMAS)
S3	64	S1 (S) S2
S4	26	RD (unique items)

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?s s1 and s2
          2818  S1
        107952  S2
     S5      90  S1 AND S2
?s s5 and (engrafable or implantable)
            90  S5
             0  ENGRAFABLE
         27655  IMPLANTABLE
     S6      2  S5 AND (ENGRAFABLE OR IMPLANTABLE)
?rd
...completed examining records
     S7      1  RD (unique items)
?t s7/3,k/all
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7/3,K/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

10114702 22081554 PMID: 12086863
Epidermal growth factor receptor and Ink4a/Arf: convergent mechanisms governing terminal differentiation and transformation along the neural stem cell to astrocyte axis.
Bachoo Robert M; Maher Elizabeth A; Ligon Keith L; Sharpless Norman E; Chan Suzanne S; You Mingjian James; Tang Yi; DeFrances Jessica; Stover Elizabeth; Weissleder Ralph; Rowitch David H; Louis David N; DePinho Ronald A
Center for Neuro-Oncology, Boston, Massachusetts 02115, USA.
Cancer cell (United States) Apr 2002, 1 (3) p269-77, ISSN 1535-6108 Journal Code: 101130617
Contract/Grant No.: P30; PHS
Document type: Journal Article
Languages: ENGLISH

Main Citation Owner: NI
Record type: Completed

Ink4a/Arf inactivation and epidermal growth factor receptor (EGFR) activation are signature lesions in high-grade *gliomas* . How these mutations mediate the biological features of these tumors is poorly understood. Here, we demonstrate that combined loss of p16(INK4a) and p19(ARF), but not of p53, p16(INK4a), or p19(ARF), enables astrocyte dedifferentiation in response to EGFR activation. Moreover, transduction of Ink4a/Arf(-/-) *neural* *stem* *cells* (NSCs) or astrocytes with constitutively active EGFR induces a common high-grade glioma phenotype. These findings identify NSCs and astrocytes as equally permissive compartments for...

... differentiation. These data support the view that dysregulation of specific genetic pathways, rather than cell-of-origin, dictates the emergence and phenotype of high-grade *gliomas*.
; Astrocytes--cytology--CY; Blotting, Western; Cells, Cultured--cytology --CY; Homozygote; Immunoenzyme Techniques; Infusion Pumps, *Implantable*; Luminescent Proteins--metabolism--ME; Magnetic Resonance Imaging; Mice; Mice, SCID; Neoplasms, Experimental--metabolism--ME; Neoplasms, Experimental--pathology--PA; Protein p53--metabolism--ME; Retroviridae --genetics--GE...
?ds

Set	Items	Description
S1	2818	((NEURAL (W) STEM (W) CELLS) OR (NEURAL (W) PROGENITORS))
S2	107952	(BRAIN (W) (TUMOR OR CANCER)) OR (GLIOBLASTOMA OR ASTROCYT-OMA OR GLIOMAS)
S3	64	S1 (S) S2
S4	26	RD (unique items)
S5	90	S1 AND S2
S6	2	S5 AND (ENGRAFABLE OR IMPLANTABLE)
S7	1	RD (unique items)

```
?s s5 and (prodrug or (cytosine (w) deaminase))
            90  S5
         17884  PRODRUG
         61391  CYTOSINE
         31488  DEAMINASE
          1530  CYTOSINE(W)DEAMINASE
     S8      9  S5 AND (PRODRUG OR (CYTOSINE (W) DEAMINASE))
?rd
...completed examining records
     S9      5  RD (unique items)
?t s9/3,k/all
```

9/3,K/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09214291 20524089 PMID: 11070094

From the cover: *neural* *stem* *cells* display extensive tropism for pathology in adult brain: evidence from intracranial *gliomas*.

Aboody K S; Brown A; Rainov N G; Bower K A; Liu S; Yang W; Small J E; Herrlinger U; Ourednik V; Black P M; Breakefield X O; Snyder E Y
Departments of Neurology, Pediatrics, and Neurosurgery, Children's Hospital, Boston, MA, USA.

Proceedings of the National Academy of Sciences of the United States of America (UNITED STATES) Nov 7 2000, 97 (23) p12846-51, ISSN 0027-8424 Journal Code: 7505876
Contract/Grant No.: CA69246; CA; NCI; CA86768; CA; NCI; HD07466; HD; NICHD; +
Comment in Proc Natl Acad Sci U S A. 2000 Nov 7;97(23) 12391-2; Comment in PMID 11070072; Comment in Proc Natl Acad Sci U S A. 2000 Nov 7;97(23):12393-5; Comment in PMID 11070073; Erratum in Proc Natl Acad Sci U S A 2001 Jan 16;98(2):777
Document type: Journal Article

Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

From the cover: *neural* *stem* *cells* display extensive tropism for pathology in adult brain: evidence from intracranial *gliomas*.

One of the impediments to the treatment of brain tumors (e.g., *gliomas*) has been the degree to which they expand, infiltrate surrounding tissue, and migrate widely into normal brain, usually rendering them "elusive" to effective resection, irradiation, chemotherapy, or gene therapy. We demonstrate that *neural* *stem* *cells* (NSCs), when implanted into experimental intracranial *gliomas* in vivo in adult rodents, distribute themselves quickly and extensively throughout the tumor bed and migrate uniquely in juxtaposition to widely expanding and aggressively advancing...

... the tumor cells (including human glioblastomas). When implanted outside the CNS intravascularly, NSCs will target an intracranial tumor. NSCs can deliver a therapeutically relevant molecule-*cytosine* *deaminase*-such that quantifiable reduction in tumor burden results. These data suggest the adjunctive use of inherently migratory NSCs as a delivery vehicle for targeting therapeutic...

Descriptors: Brain--pathology--PA; *Brain Neoplasms--pathology--PA; * *Glioblastoma*--pathology--PA; *Neurons--physiology--PH; *Nucleoside Deaminases--genetics--GE; *Stem Cells--physiology--PH; Brain Neoplasms --therapy--TH; Cell Movement--physiology--PH; Disease Models, Animal; Gene Therapy--methods--MT; *Glioblastoma*--therapy--TH; Hematopoietic Stem Cell Transplantation; Mice; Mice, Nude; Neurons--cytology--CY; Rats; Rats, Inbred F344; Stem Cells--cytology--CY; Tropism

Enzyme No.: EC 3.5.4 (Nucleoside Deaminases); EC 3.5.4.1 (*cytosine* *deaminase*)

Chemical Name: Nucleoside Deaminases; *cytosine* *deaminase*

9/3,K/2 (Item 2 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

09214206 20524003 PMID: 11070073

Can *neural* *stem* *cells* be used to track down and destroy migratory *brain* *tumor* cells while also providing a means of repairing tumor-associated damage?

Noble M

Center for Cancer Biology, University of Rochester Medical Center, 601 Elmwood Avenue, Box 633, Rochester, NY 14642, USA.

Proceedings of the National Academy of Sciences of the United States of America (UNITED STATES) Nov 7 2000, 97 (23) p12393-5, ISSN 0027-8424 Journal Code: 7505876

Comment on Proc Natl Acad Sci U S A. 2000 Nov 7;97(23) 12846-51; Comment on PMID 11070094

Document type: Comment; Journal Article; Review; Review, Tutorial

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Can *neural* *stem* *cells* be used to track down and destroy migratory *brain* *tumor* cells while also providing a means of repairing tumor-associated damage?

Descriptors: Brain Neoplasms--therapy--TH; *Cell Movement--physiology--PH ; **Glioblastoma*--therapy--TH; *Hematopoietic Stem Cell Transplantation; *Neurons--physiology--PH; *Stem Cells--physiology--PH

Enzyme No.: EC 3.5.4 (Nucleoside Deaminases); EC 3.5.4.1 (*cytosine* *deaminase*)

Chemical Name: Prodrugs; Flucytosine; Fluorouracil; Nucleoside Deaminases ; *cytosine* *deaminase*

9/3,K/3 (Item 1 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

12800498 BIOSIS NO.: 200100007647

***Neural* *stem* *cells* display extensive tropism for pathology in adult brain: Evidence from intracranial *gliomas*.**

AUTHOR: Aboody Karen S; Brown Alice; Rainov Nikolai G; Bower Kate A; Liu Shaoxiong; Yang Wendy; Small Juan E; Herrlinger Ulrich; Ourednik Vaclav; Black Peter McL; Breakefield Xandra O; Snyder Evan Y(a)
AUTHOR ADDRESS: (a)Departments of Neurology, Pediatrics, and Neurosurgery, Children's Hospital, Harvard Medical School, Boston, MA, 02115: Snyder@A1.TCH.Harvard.edu**USA

JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 97 (23):p12846-12851 November 7, 2000
MEDIUM: print
ISSN: 0027-8424
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

***Neural* *stem* *cells* display extensive tropism for pathology in adult brain: Evidence from intracranial *gliomas*.**

ABSTRACT: One of the impediments to the treatment of brain tumors (e.g., *gliomas*) has been the degree to which they expand, infiltrate surrounding tissue, and migrate widely into normal brain, usually rendering them "elusive" to effective resection, irradiation, chemotherapy, or gene therapy. We demonstrate that *neural* *stem* *cells* (NSCs), when implanted into experimental intracranial *gliomas* in vivo in adult rodents, distribute themselves quickly and extensively throughout the tumor bed and migrate uniquely in juxtaposition to widely expanding and aggressively advancing...

...the tumor cells (including human glioblastomas). When implanted outside the CNS intravascularly, NSCs will target an intracranial tumor. NSCs can deliver a therapeutically relevant molecule-*cytosine* *deaminase*-such that quantifiable reduction in tumor burden results. These data suggest the adjunctive use of inherently migratory NSCs as a delivery vehicle for targeting therapeutic...

DESCRIPTORS:
CHEMICALS & BIOCHEMICALS: *cytosine* *deaminase* cDNA...

9/3,K/4 (Item 2 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

12307437 BIOSIS NO.: 200000065304

A novel platform for gene therapy against brain tumors: Foreign gene expressing *neural* *stem* *cells* (NSCs) display tropism for intracranial *gliomas*.

AUTHOR: Aboody Karen S(a); Rainov Nikolai G; Bower Kate A(a); Small Juan E (a); Liu Shaoxiong(a); Brown Alice; McL Black Peter; Breakefield Xandra O ; Snyder Evan Y(a)
AUTHOR ADDRESS: (a)Depts of Neurology, Pediatrics, and Neurosurgery, Children's Hospital, Harvard Med. School, Boston, MA**USA

JOURNAL: Society for Neuroscience Abstracts 25 (1-2):p246 1999
CONFERENCE/MEETING: 29th Annual Meeting of the Society for Neuroscience, Part 1 Miami Beach, Florida, USA October 23-28, 1999
SPONSOR: The Society for Neuroscience
ISSN: 0190-5295
RECORD TYPE: Citation
LANGUAGE: English

A novel platform for gene therapy against brain tumors: Foreign gene expressing *neural* *stem* *cells* (NSCs) display tropism for intracranial *gliomas*.

...REGISTRY NUMBERS: *CYTOSINE* *DEAMINASE*

DESCRIPTORS:
  ORGANISMS: PARTS ETC: *neural* *stem* *cells*--
  CHEMICALS & BIOCHEMICALS: *cytosine* *deaminase*

9/3,K/5 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE
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11621212 EMBASE No: 2002193703
Global gene and cell replacement strategies via stem cells
Park K.I.; Ourednik J.; Ourednik V.; Taylor R.M.; Aboody K.S.; Auguste K.I.; Lachyankar M.B.; Redmond D.E.; Snyder E.Y.
E.Y. Snyder, Harvard Medical School, Harvard Institutes of Medicine, Department of Neurology, 77 Avenue Louis Pasteur, Boston, MA 02115 United States
AUTHOR EMAIL: esnyder1@caregroup.harvard.edu
Gene Therapy (GENE THER.) (United Kingdom) 2002, 9/10 (613-624)
CODEN: GETHE ISSN: 0969-7128
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 67

The inherent biology of *neural* *stem* *cells* (NSCs) endows them with capabilities that not only circumvent many of the limitations of other gene transfer vehicles, but that enable a variety of novel...
DRUG DESCRIPTORS:
drug vehicle; intrinsic factor--endogenous compound--ec; neurotrophin 3 --drug therapy--dt; neurotrophin 3--pharmaceutics--pr; neurotrophin 3 --pharmacology--pd; *cytosine* *deaminase*--drug therapy--dt; *cytosine* *deaminase*--pharmaceutics--pr; *cytosine* *deaminase*--pharmacology--pd
MEDICAL DESCRIPTORS:
...gene expression; signal transduction; neuroprotection; nerve regeneration; brain ischemia--drug therapy--dt; brain ischemia--therapy--th ; brain hypoxia--drug therapy--dt; brain hypoxia--therapy--th; *brain* *tumor*--drug therapy--dt; *brain* *tumor*--therapy--th; Alzheimer disease --therapy--th; human; nonhuman; mouse; rat; controlled study; animal tissue ; animal cell; review; priority journal
CAS REGISTRY NO.: 9025-05-2 (*cytosine* *deaminase*)
?ds

Set	Items	Description
S1	2818	((NEURAL (W) STEM (W) CELLS) OR (NEURAL (W) PROGENITORS))
S2	107952	(BRAIN (W) (TUMOR OR CANCER)) OR (GLIOBLASTOMA OR ASTROCYT-OMA OR GLIOMAS)
S3	64	S1 (S) S2
S4	26	RD (unique items)
S5	90	S1 AND S2
S6	2	S5 AND (ENGRAFABLE OR IMPLANTABLE)
S7	1	RD (unique items)
S8	9	S5 AND (PRODRUG OR (CYTOSINE (W) DEAMINASE))
S9	5	RD (unique items)

?logoff
06oct03 15:18:36 User259876 Session D553.2
$2.29 0.717 DialUnits File155
$4.41 21 Type(s) in Format 3
$4.41 21 Types
$6.70 Estimated cost File155
$1.30 0.439 DialUnits File159
$1.30 Estimated cost File159
$3.98 0.710 DialUnits File5
$12.25 7 Type(s) in Format 3
$12.25 7 Types
$16.23 Estimated cost File5
$6.11 0.661 DialUnits File73
$10.20 4 Type(s) in Format 3
$10.20 4 Types

$16.31 Estimated cos[redacted]File73
OneSearch, 4 Files, 2.527 DialUnits FileOS
$1.16 TELNET
$41.70 Estimated cost this search
$42.09 Estimated total session cost 2.618 DialUnits

Status: Signed Off. (5 minutes)